Local sweating and cutaneous blood flow during exercise in hypobaric environments

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KOLKA, MARGARET A., LOU A. STEPHENSON, PAUL B. ROCK, AND RICHARD R. GONZALEZ. *Local sweating and cutaneous blood flow during exercise in hypobaric environments.* J. Appl. Physiol. 62(6): 2224–2229, 1987.—The effect of acute hypobaric hypoxia on local sweating and cutaneous blood flow was studied in four men and four women (follicular phase of menstrual cycle), who exercised at 60% of their altitude-specific peak aerobic power for 35 min at barometric pressures (PB) of 770 Torr (sea level), 552 Torr (2,596 m), and 428 Torr (4,575 m) at an ambient temperature of 30°C. We measured esophageal temperature (T_{es}), mean skin temperature ($\bar{T}_{sk}$, 8 sites), and local sweating ($\dot{m}_s$) from dew-point sensors attached to the skin at the chest, arm, and thigh. Skin blood flow (SkBF) of the forearm was measured once each minute by venous occlusion plethysmography. There were no gender differences in the sensitivity (slope) or the threshold of either $\dot{m}_s/T_{es}$ or SkBF/T_{es} at any altitude. No change in the T_{es} for sweating onset occurred with altitude. The mean slopes of the $\dot{m}_s/T_{es}$ relationships for the three regional sites decreased with increasing altitude, although these differences were not significant between the two lower PBs. The slope of SkBF/T_{es} was reduced in five of the eight subjects at 428 Torr. Enhanced body cooling as a response to the higher evaporative capacity of the environment is suggested as a component of these peripheral changes occurring in hypobaric hypoxia.

altitude; dew point; esophageal temperature; gender; local sweating; skin blood flow

IN HYPOBARIC ENVIRONMENTS, the maximal evaporative capacity (E_{max}) is enhanced due to an increase in the effective mass transfer coefficient for any given air movement (3, 21). In theory, at a given relative exercise intensity, the relative evaporation of a given volume of sweat is greater at a lower barometric pressure. Because evaporation is greater, there is a lower skin wettedness [ratio of evaporation from the skin (E_{sk}) to E_{max}] at a lower barometric pressure (PB). However, at higher altitudes, the increased insensible heat loss (evaporation) leads to a lower core and skin temperature. This effect results in depressed thermoregulatory sweating at altitude. Additionally, the lower core and skin temperatures may attenuate blood flow to the skin.

In lowlanders unacclimated to altitude, chronic altitude exposure (3 wk) to 474 Torr resulted in a higher rectal temperature threshold for the onset of regulatory sweating with no change in the sweating sensitivity (i.e., mg·cm^{-2}·min^{-1} per core temperature change) (22). However, this delayed onset of sweating may have resulted from chronic altitude-induced dehydration (13). During simulated altitude exposure (596 and 462 Torr), higher whole-body evaporative heat loss (E_{tot}) was observed in subjects exercising at the same absolute exercise intensity as sea level (11). The increase in E_{tot} observed was entirely from an increase in respiratory water loss. E_{sk} was unchanged by altitude. That design required individuals to work at a higher percentage of their peak O_2 requirements at the depressed PB; however, no change in E_{sk} was apparent at either altitude (11).

The hypoxia that occurs in hypobaric environments is also a factor in control of thermoregulatory sweating and skin blood flow (SkBF) in simulated altitude studies. In one study, the short-term regulation of SkBF and body temperature during moderate exercise was not changed by breathing a hypoxic gas mixture (23). Wagner et al. (29) have suggested reduced blood flow from core to skin at 446 Torr but did not discuss possible changes in the control of skin blood flow.

A rigorous evaluation of the control of thermoregulatory sweating and cutaneous blood flow during acute hypobaria has not yet been undertaken. The cutaneous vascular response to hypobaria has not been adequately characterized, because previous studies have used indirect indexes such as skin temperature measurements to estimate cutaneous blood flow response or hypoxic breathing to simulate the hypoxia of altitude. Furthermore, the evaluation of weight loss data from before and after an experiment provides very limited information regarding the control of regulatory sweating at reduced PB because of the interaction of several thermal inputs superimposed at the same time. In the present study, we examined the transient responses of sweating ($\dot{m}_s$) and SkBF to esophageal temperature (T_{es}) elevation induced by exercise during acute altitude exposure. We hypothesized that the increased effective mass transfer that occurs in hypobaria would affect thermoregulation. Specifically, we were interested in determining whether there was a change in the esophageal temperature threshold for the initiation of thermoregulatory sweating and/or cutaneous vasodilation and/or the sensitivity of the thermoregulatory effectors to a given internal thermal drive at a constant ambient temperature.

METHODS

Eight subjects (4 men, 4 women) participated in the experiments after giving their informed consent. The subjects had an average age of 26 ± 4 (SD) yr, weight of

71.1 ± 7.9 kg, and DuBois surface area of 1.82 ± 0.13 m^2. All subjects were accustomed to exercise and measurement routines. Female subjects were tested during the follicular phase (*days 1–9*) of the menstrual cycle to control for thermoregulatory differences that occur between the follicular and luteal phases as established by previous studies (26). Subjects exercised in a seated position behind a modified cycle ergometer. We determined peak aerobic power ($\dot{V}O_{2\,peak}$) for each subject in this position (16). Separate maximal exercise tests were made at 770 Torr (sea level), 552 Torr [moderate altitude (AI), 2,596 m], and 428 Torr [high altitude (AII), 4,575 m] in a hypobaric chamber (ambient temperature = 24°C, dew-point temperature = 10°C). O_2 consumption by standard open-circuit techniques was calculated on-line with a Hewlett-Packard (HP) computer modified for hypobaric environments. The $\dot{V}O_{2\,peak}$ was defined as the maximal O_2 uptake that occurred when increases in work load did not produce a subsequent increase in $\dot{V}O_2$. The order of sea level and altitude $\dot{V}O_{2\,peak}$ tests was randomized, and subjects were not advised of the specific altitude sojourn during testing. The mean sea level $\dot{V}O_{2\,peak}$ was 2.92 ± 0.68 (SD) l/min, 2.68 ± 0.56 l/min at 552 Torr, and 2.36 ± 0.46 l/min at 428 Torr, which are normal decreases for the reduced PB.

Experimental procedures. Subjects reported to the hypobaric chamber between 0800 and 1200 h after a light breakfast. Each person was tested at the same time of day during all exposures to control for the circadian variation in heat loss responses (27). They were dressed in shorts, socks, shoes, and 100% cotton surgical scrub shirts. Submaximal exercise was performed once by each subject at each altitude. The environmental temperature (T_a) was 30°C and the ambient dew point (T_{dp}) was held constant at 10°C (partial pressure of water vapor = 1.2 kPa) during all experiments. The mean work load was 101 ± 30 (SD) W at sea level, 95 ± 21 W at AI, and 84 ± 18 at AII, which averaged 60% of the $\dot{V}O_{2\,peak}$ for each subject at each altitude. The order of environmental conditions was randomized, and total time at any altitude during an experiment was <1.25 h.

Subjects rested for 10 min after instrumentation and achievement of thermal equilibration [defined by changes in T_{es} and mean skin temperature ($\bar{T}_{sk}$) of ≤± 0.1°C over a 30-min period]. A bout of 35 min of semisupine cycle exercise then followed at the prescribed altitude. Continuous measurements of T_{es}, T_{sk} from eight sites (20), local skin temperature ($T_{a,l}$) adjacent to the dew-point sensors, and $\dot{m}_s$ from the upper arm, chest, and thigh (10) from ventilated dew-point sensors were recorded on a HP85 computer. The dew-point sensors were ventilated with ambient air from the chamber. The airflow to the sensors was calibrated in situ at each specific altitude and corrected for density and viscosity. Sweating rates were calculated as described previously (10) as

$$\dot{m}_s = (\Delta P_{dp})(AF)/(R_w \cdot A \cdot T) \cdot 1{,}000 \quad [mg \cdot cm^{-2} \cdot min^{-1}]$$

where ΔP_{dp} is the water vapor pressure gradient between inlet air and the water vapor pressure as measured by the dew-point sensor; R_w is the gas constant for water vapor (0.0821 $atm \cdot m^3 \cdot g^{-1} \cdot mol^{-1} \cdot K^{-1}$); A is the area of the sweat capsule enclosing the skin site (cm^{-2}); T is the absolute temperature (°K) of ambient air entering the capsule; and AF is the air flowing through the capsule (m^3/min). Local sweating rates can be measured with an accuracy of 0.05 $mg \cdot cm^{-2} \cdot min^{-1}$ with this system. Forearm blood flow was calculated each minute by venous occlusion plethysmography, which is based on changes in limb girth (14, 31). Metabolic heat production was evaluated at rest and during steady-state exercise (final 15 min). Total body sweating rate was evaluated from changes in body (taking into account clothing weight) weight corrected for convective and evaporative heat loss from the respiratory tract (5, 9).

Statistical analysis. The T_{es} thresholds for sweating and SkBF were calculated for each experiment by analyzing the inflection points of the exercise transient phase for each specific effector response to T_{es}. The exercise transient phase is defined as the time of exercise during which a rapid increase in T_{es}, sweating rate above that owing to skin diffusion (insensible water loss), and increased SkBF are observed. A regression equation was calculated for each subject during the exercise transient for $\dot{m}_s/T_{es}$ and $SkBF/T_{es}$. The data collected after T_{es} reached a steady level were not included in the calculation of either linear regression equation. The T_{es} threshold for sweating was calculated from the regression equation at $\dot{m}_s = 0.06\ mg \cdot cm^{-2} \cdot min^{-1}$. The T_{es} threshold for arm vasodilation is defined as the point where the regression equation intersects the resting SkBF.

Analysis of variance was performed on all data by using the individual slopes and thresholds of both $\dot{m}_s/T_{es}$ and $SkBF/T_{es}$. All steady-state data were also analyzed by repeated analysis of variance. Post hoc tests (Tukey) were performed whenever a significant F ratio occurred ($P < 0.05$). Data in RESULTS are presented as means ± SD.

RESULTS

A typical experimental time course demonstrating the transient periods for T_{es} and chest $\dot{m}_s$ is shown in Fig. 1, *A–C*, for a single subject. The transient increase in T_{es} and chest $\dot{m}_s$ is apparent shortly after the initiation of exercise. No gender differences were found in either the sensitivity (slope) or the threshold of either $\dot{m}_s/T_{es}$ or $SkBF/T_{es}$ for any measured skin site at any altitude; therefore all data presented will be the mean of all eight subjects.

Table 1 shows the T_{es} thresholds and slopes for $\dot{m}_s$. The T_{es} threshold for sweating was not affected by altitude. The onset of arm sweating always occurred at a higher core temperature than that of either chest or thigh. Figure 2 is a composite profile of $\dot{m}_s/T_{es}$ for each hypobaric environment in a representative subject during the exercise transient. The slopes (sensitivity to core temperature drive) of $\dot{m}_s$ show a clear decrease at increased altitude. For the whole group, sensitivity of chest sweating was depressed an average of 26% ($P < 0.05$) at moderate altitude and 38% ($P < 0.05$) at high altitude. Arm and thigh sensitivities ($\dot{m}_s$) were reduced an average of 44 and 17% ($P < 0.05$), respectively, for both moderate and high altitude. No effect of altitude on whole-body sweating, as measured by weight loss pre- and postex-

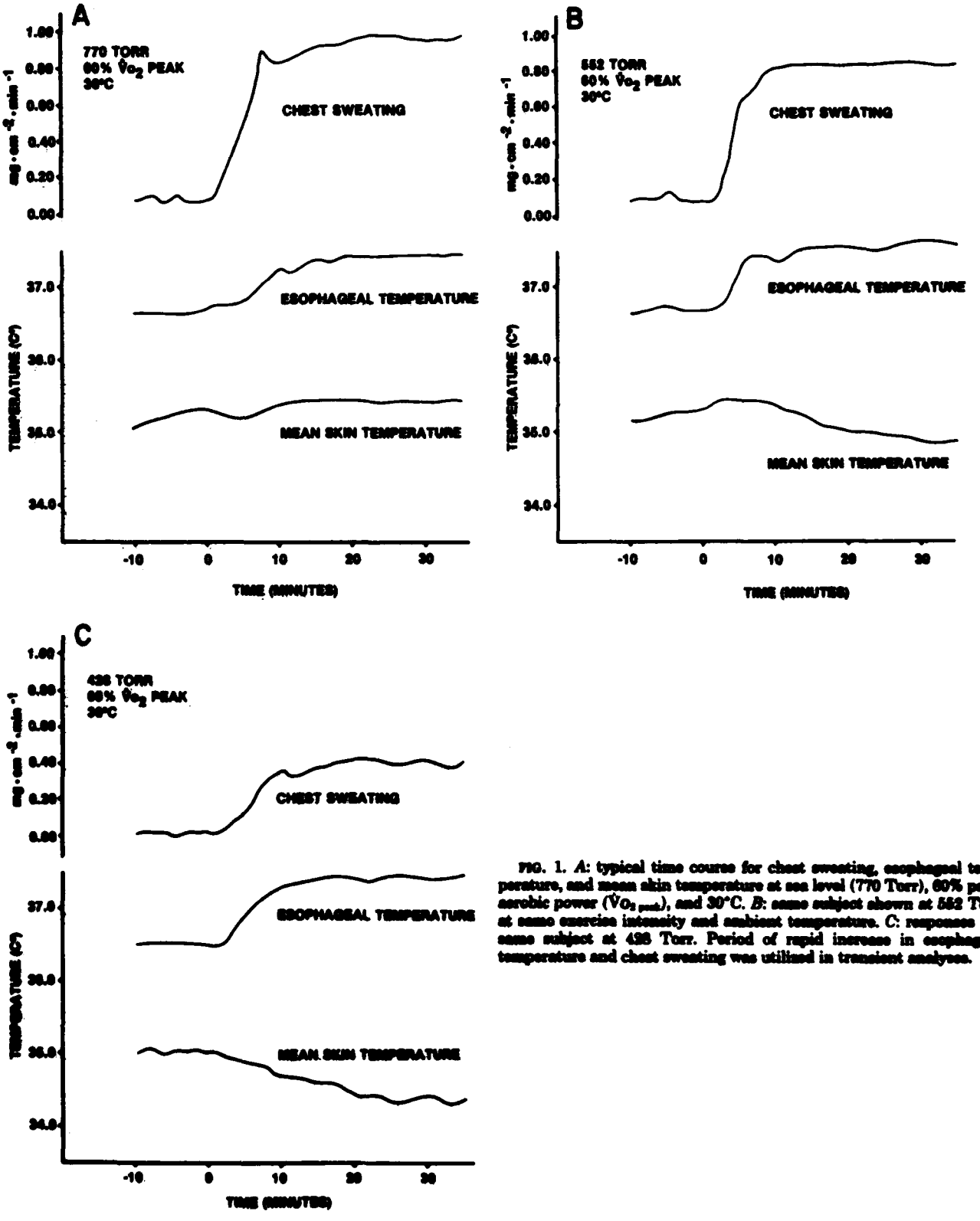

FIG. 1. *A*: typical time course for chest sweating, esophageal temperature, and mean skin temperature at sea level (770 Torr), 60% peak aerobic power ($\dot{V}O_{2\,peak}$), and 30°C. *B*: same subject shown at 552 Torr at same exercise intensity and ambient temperature. *C*: responses for same subject at 428 Torr. Period of rapid increase in esophageal temperature and chest sweating was utilized in transient analyses.

posure, was apparent during the moderate exercise at the three levels of simulated altitude.

In three of the eight subjects, there was a very high level of local skin vasoconstrictor activity during the early exercise transients at reduced P_B due to low forearm and mean skin temperatures. This prevented full data analysis of the $SkBF/T_{es}$ relationship for these subjects. The data from these three subjects were not included in the regression analysis, not due to nonconforming data but due to very low SkBF during the core temperature transient at the beginning of exercise (30). Therefore, only data on five subjects were used in the

TABLE 1. *Esophageal temperature thresholds for sweating and slopes of linear regression equation generated from transient response of $\dot{m}_s/T_{es}$*

	Sea Level	AI	AII
Chest			
T_{es}, °C	36.62±0.21	36.73±0.29	36.66±0.21
$\dot{m}_s/T_{es}$	0.87±0.28	0.64±0.21*	0.54±0.19*
Arm			
T_{es}, °C	36.74±0.22	36.83±0.24	36.83±0.23
$\dot{m}_s/T_{es}$	1.06±0.48	0.60±0.22*	0.59±0.24*
Thigh			
T_{es}, °C	36.45±0.24	36.64±0.27	36.58±0.10
$\dot{m}_s/T_{es}$	0.96±0.65	0.81±0.20*	0.81±0.41*

Values are means ± SD. AI, moderate altitude; AII, high altitude; $\dot{m}_s$, sweat rate; T_{es}, esophageal temperature measured in °C. * Different from sea-level value, $P < 0.05$.

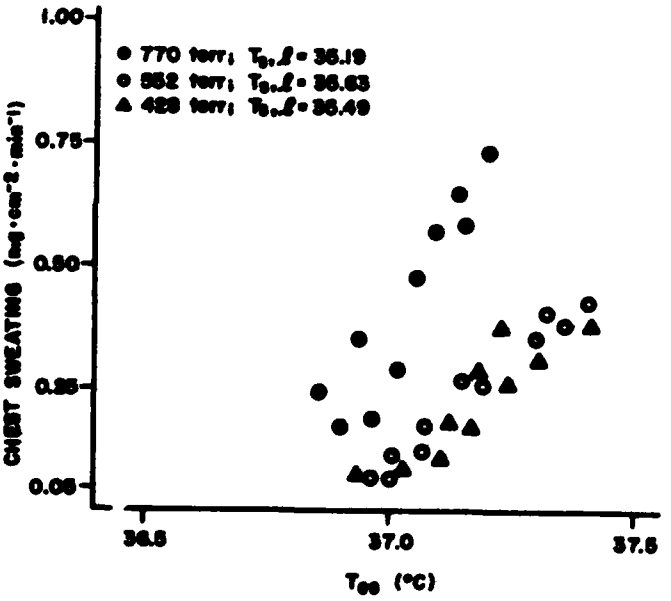

FIG. 2. Chest sweating plotted against esophageal temperature (T_{es}) during transient period of these variables during 60% peak aerobic power ($\dot{V}O_{2\ peak}$) exercise at 30°C at 3 altitudes for a representative subject. T_{sk}, local skin temperature of chest adjacent to dew-point sensor. At sea level $r = 0.98$, $m = 1.27$, $b = -46.33$; at 552 Torr $r = 0.96$, $m = 0.92$, $b = -33.56$; at 428 Torr $r = 0.96$, $m = 0.46$, $b = -16.69$.

TABLE 2. *Mean slopes of SkBF and T_{es} for vasodilation (T_a = 30°C)*

	n	Sea Level	AI	AII
Slope, ml·100 ml^{-1}·min^{-1}	5	6.5±2.3	6.1±2.7	5.8±4.4*
T_{es}, °C	5	36.6±0.07	36.6±0.2	36.3±0.2*

Values are means ± SD; n = 5 subjects. SkBF, skin blood flow; T_{es}, esophageal temperature; T_a, ambient temperature; AI, moderate altitude; AII, high altitude. * Different from sea-level value, $P < 0.05$.

statistical presentation of the data. The mean of five subjects is presented in Table 2 as the slope and T_{es} onset at each of the three PB's. There was a significant depression (11%) in the slope and a decrease in the T_{es} (−0.3°C) at the onset of arm vasodilation in the higher altitude (AII) experiments.

Mean skin temperature (Tables 3 and 4) was not significantly different during the exercise transient period at the three altitudes but was lower during steady-state exercise at both AI and AII. T_{sk} for the three skin

TABLE 3. *Local skin temperature and T_{sk} averaged across exercise transients, and change in T_{arm} and T_{sk} from regression analysis during transient period*

Temperature	Sea Level	AI	AII
Chest, °C	34.90±1.79	34.85±1.03	35.03±0.30
Arm, °C	34.72±0.70	33.83±0.77	34.37±0.51
Thigh, °C	34.53±0.60	33.78±0.95	33.79±0.37
Mean skin, °C	34.65±0.56	33.98±0.67	34.08±0.21
dT_{arm}/dt, °C/min	−0.025±0.04	0.025±0.09	−0.080±0.09*
dT_{sk}/dt, °C/min	−0.022±0.03	−0.035±0.04	−0.055±0.05†

Values are means ± SD. AI, moderate altitude; AII, high altitude; dT_{arm}/dt, change in local arm temperature over time; dT_{sk}/dt, change in mean skin temperature over time. * $P = 0.06$; † $P = 0.11$.

TABLE 4. *Mean steady-state data*

	Sea Level	AI	AII
T_{es}, °C	37.45±0.23	37.43±0.27	37.14±0.30*†
ΔT_{es}, °C	0.59±0.22	0.65±0.15	0.61±0.18
T_{sk}, °C	34.27±0.68	33.64±0.43	33.13±0.76*
$\dot{m}_{s,arm}$, mg·cm^{-2}·min^{-1}	1.29±0.40	0.92±0.34	0.75±0.18*
SkBF, ml·100 ml^{-1}·min^{-1}	8.53±3.37	7.27±3.65	5.99±2.70
$T_{s,arm}$, °C	33.74±0.81	33.95±0.77	32.96±0.81†
Weight loss, g/min^{-1}	10.14±2.47	11.06±3.03	9.39±1.91
Metabolism, W/m^2	307±61	309±59	263±38*†

Values are means ± SD. AI, moderate altitude; AII, high altitude; T_{es}, esophageal temperature; ΔT_{es}, change in esophageal temperature from rest to steady-state exercise; $\dot{m}_{s,arm}$, sweat rate of arm; SkBF, skin blood flow; $T_{s,arm}$, temperature of arm. * Different from sea-level values, $P < 0.05$. † Different from AI values, $P < 0.05$.

locations near the dew-point sensors is also shown in Table 3. The mean steady-state thermoregulatory parameters are shown in Table 4 for the eight subjects of the study. T_{es}, T_{sk}, arm sweating ($T_{s,l}$), and metabolic heat production were all significantly lower at AII compared with the responses evident at sea level. There were no significant differences in the steady-state SkBF at any given altitude.

DISCUSSION

In this study we controlled the work intensity at each altitude so that there was a consistent thermoregulatory perturbation (25), thereby allowing a more direct comparison of the effects of hypobaric environments on thermoregulatory sweating and SkBF. Characteristically, sweating and peripheral blood flow have been shown to respond in a proportional manner to changes in the thermal controller (12, 18, 19, 23–25). Therefore, we used a relative exercise intensity (60% altitude specific $\dot{V}O_{2\ peak}$) as an endogenous thermal clamp to limit the change in T_{es} input to the thermal controller (19, 25). The change in body temperature was similar at all altitudes even though resting core temperature was lower at AII in all subjects (Table 4). This change resulted in T_{es} increases from rest to steady-state exercise of 0.5, 0.6, and 0.6°C at sea level, moderate, and high altitude, respectively. By matching the internal thermal drive at each PB, a suppression in the sensitivity of the relationship of local sweating or cutaneous vasodilation to core temperature was observed at both moderate and high altitude.

The decreases in the slope of $\dot{m}_s/T_{es}$ and SkBF/T_{es} during acute exposure to moderate and high altitude

appear to be a result of local alterations of the sweat gland and/or peripheral vasculature (12, 18). These modifications may be caused by local skin influences, because the temperatures were lower at AII but not AI during steady-state exercise. The local skin temperature influences on the peripheral sweating mechanism are well documented and have been discussed in greater detail (1, 2, 4, 18). The drop in arm $\bar{T}_{sk}$ with exercise ($d\bar{T}_{loc}/dt$) was more rapid at high altitude (Table 3) over the transient period of 5–7 min and may be one source of the depressed thermal sensitivity. Other physical influences, such as changes in the liquid-vapor interfaces within the skin itself and/or biochemical changes at the level of the sweat gland may also be a source of the depressed thermal sensitivity and warrant further study.

The continuous measurement of local sweating allowed an evaluation of thermoregulatory control of sweating that was not possible by evaluating body weight changes merely from pre- and postexposure. The higher skin diffusion at reduced PB decreased the requirement for regulatory sweating, as evidenced by our lower resting core temperatures at AII. Pre- and postexposure weight changes cannot give any indication about changes in the control of regulatory sweating at altitude as the heat content of the body is increasing.

Greenleaf and colleagues (11) showed an increase in whole-body evaporative heat loss at altitude in subjects exercising at the same work load as sea level. This increase was singularly related to an increase in the respiratory water loss. Water loss through sweating was unchanged at both 596 and 462 Torr even though subjects worked harder at altitude (65% $\dot{V}O_{2\,max}$) than at sea level (45% $\dot{V}O_{2\,max}$). These authors (11) were not able to document depressed sweating sensitivity at altitude as they did not evaluate the exercise transient.

The effect of the relative hypoxia at the sweat gland itself during AI and AII was also considered. Elizondo (4) suggested that a lowered O_2 tension after arterial occlusion possibly affects synthesis of transmitter substance (i.e., acetylcholine), thereby reducing synaptic transmission. When physostigmine, an anticholinesterase, was administered in combination with arterial occlusion no change in sweating occurred (4) confirming that reduced transmission was due to lack of neurotransmitter substance. At an altitude of 428 Torr, arterial O_2 saturation is depressed but not to the low levels induced by arterial occlusion. If local sweat gland activity were depressed solely by a hypoxic influence, the gain of $\dot{m}_s/T_{es}$ would be reduced as was apparent in our study; however, core temperature would reach a higher steady-state level due to compromised evaporative heat loss. Although the slope of $\dot{m}_s/T_{es}$ was reduced at altitude (Fig. 2), there was no difference in the change in T_{es} at steady state in the present study (Table 4). Thus the results of our study do not support a local hypoxic inhibition of sweating.

In the present study, no change in the core temperature threshold for sweating (Table 1) was observed with acute altitude exposure. Thus our results fail to demonstrate a central alteration (12) in the control of sweating during exercise in the presence of acute hypobaric hypoxia as has been shown after chronic altitude exposure (22). Because the increase in body temperature during exercise was similar at all altitudes, one might suggest that the central thermal controller did, in fact, sum the signals from core to skin drives equally at all altitudes (PB). The disparate results between our study and that seen with one chronic altitude study could relate to those subjects in the chronic exposure possibly being dehydrated (13). This could account for the upward threshold displacement or delay in heat dissipation mechanisms (28).

The depressed sensitivity (gain) in $\dot{m}_s/T_{es}$ from sweating measurements in the chest compared with the slope changes evident from extremity locations evaluated in our study conflicts with other observations (18) in which a higher gain and a lower onset T_{es} temperature occurred in the chest $\dot{m}_s/T_{es}$ relationship compared with more peripheral sites. However, subjects in the present study wore surgical scrub shirts and were seated in a chair behind the ergometer, thereby creating a different local environment at the chest, which may have influenced the local evaporative heat transfer at each site (5, 8). We also measured dew-point changes directly at the skin. The microenvironment at the chest (under a layer of cloth) had a higher water vapor pressure than at the thigh, which was manifested in the different local skin wettedness and heat transfer coefficients at the two sites. Local chest wettedness (0.72 at AII), measured with an unventilated dew-point sensor, was ~40% higher than thigh (0.50) and arm (0.52) skin wettedness as determined by independent measurements of skin saturation pressure to ambient water pressure differences (6). Thigh and arm mass transfer coefficients were higher than those of the chest (7) enabling more effective evaporation and lowered skin wettedness. Yet, all three sites used for local sweating measurements exhibited the depression in sensitivity with reduced PB (Table 1); thus even with the confounding effect of the clothing worn, all sites showed the effect of the hypobaria.

We observed a great variability among our subjects in the measurement of forearm cutaneous blood flow in these experiments at all altitudes. The low slopes seen are related to the low mean skin and local skin (forearm) temperatures (17). Three of our subjects did not vasodilate until late in the exercise transient at high altitude when T_{es} rose accordingly, and this was due to a high level of vasoconstrictor tone resulting from the low skin temperature. However, these results also support our contention that the sensitivity of cutaneous vasodilation is suppressed at high altitude as our subjects' core temperature increased without a clearly measurable concomitant change in cutaneous blood flow.

Table 2 also shows a lower T_{es} for vasodilatory onset at high altitude. This response is in contrast to our local sweating data and lends credence to a possible central nervous system alteration in the control of cutaneous blood flow in concert with local peripheral changes. Rowell and colleagues (23), in a hypoxic breathing study, failed to show any alteration in cutaneous blood flow to T_{es} compared with breathing normal room air. However, the different heat transfer characteristics of a total ambient hypobaric challenge input differently on the peripheral thermal receptors than when breathing hypoxic gas mixtures at sea level. Wenger et al. (30) have recently reported that a 1°C change in skin temperature (such as

we report at steady state) is sufficient to reduce the slope of the blood flow-to-T_{es} relationship by 12–13%, a response very comparable with our data. In addition, the higher circulating catecholamines seen at altitude at the same relative percent $\dot{V}O_{2\,peak}$ (15) could contribute to the observed reduction in forearm blood flow.

In summary, the effects of hypobaric hypoxia on heat exchange were evaluated without confounding inputs from disparate levels of core temperature change between altitudes. A depression in the thermal sensitivity of both local sweating and forearm cutaneous blood flow was observed at both moderate and high altitude. There were no changes in the sweating threshold with acute moderate and high altitude; however, the vasodilatory threshold was lowered at reduced PB.

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